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The role of premorbid psychological attributes in short- and long-term adjustment after cardiac disease. A prospective study in the elderly in The Netherlands

Cornelia H.M. van Jaarsveld[a,*], Adelita V. Ranchor[a], Robbert Sanderman[a], Johan Ormel[b], Gertrudis I.J.M. Kempen[c]

[a]*Department of Public Health and Health Psychology, Northern Centre for Healthcare Research, University of Groningen, P.O. Box 196, 9700 AD Groningen, The Netherlands*

[b]*Department of Social Psychiatry, University of Groningen, P.O. Box 30.001, 9700 RB Groningen, The Netherlands*

[c]*Section of Medical Sociology, Health Care Studies, Maastricht University, P.O. Box 616, 6200 MD Maastricht, The Netherlands*

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Abstract

The role of mastery, self-efficacy expectancies and neuroticism in explaining individual differences in physical and psychological adjustment to cardiac disease was studied in 208 patients. Premorbid data were available from a community-based survey in the Netherlands. Hierarchical linear regression analyses showed that self-efficacy expectancies at baseline were significantly related to adjustment in terms of physical functioning in the short- and long-term and depressive symptoms in the short-term (six weeks after diagnosis). Mastery was significantly related to depressive symptoms and anxiety in the long-term (1 year after diagnosis). Neuroticism was a predictor for depressive symptoms and anxiety both in the short- and long-term. The results of this longitudinal study showed that premorbidly assessed psychological attributes do have a role in explaining individual differences in vulnerability to negative consequences of cardiac disease.

Keywords: Psychological attributes; Mastery; Self-efficacy expectancies; Neuroticism; Cardiac disease; Adjustment; The Netherlands

Introduction

The impact of cardiac disease on patients' well-being, physical and psychological functioning is profound and has been shown in several cross-sectional studies (Jaarsma et al., 1999; Stewart et al., 1989; Verbrugge & Patrick, 1995), and longitudinal studies (Jaarsveld, Sanderman, Miedema, Ranchor, & Kempen, 2001; Ladwig, Roll, Breithardt, Budde, & Borggrefe, 1994; Shnek, Irvine, Stewart, & Abbey, 2001). Cardiac diseases, such as congestive heart failure (CHF) and acute myocardial infarction (AMI) may affect people's life both in the short- and the long-term. In a previous report on the sample presented in this paper, an immediate increase in physical dysfunctioning and anxiety was described and a delayed but also significant increase in depressive symptoms at six months after the diagnosis of cardiac disease, which persisted up to one year after diagnosis. Although mean scores at the group level showed these effects clearly, large individual differences in functioning at follow-up were observed (Jaarsveld et al., 2001).

*Corresponding author. Tel.: +31-50-3633208; fax: +31-50-363-6251.

E-mail address: e.van.jaarsveld@med.rug.nl (C.H.M. van Jaarsveld).

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This heterogeneity in adjustment is not entirely clear. The literature has generally failed to show that clinical parameters of cardiac disease severity predict adjustment (Frasure-Smith, Lesperance, & Talaji, 1993; Sullivan et al., 1996). Maladjustment may result from a combination of factors, including family history, biological markers, experience of damaging circumstances, sociodemographic characteristics or personality and behavioral attributes. In other words, each individual copes differently with stressful situations, depending on their personal attributes. The present study focuses on the role of psychological attributes, more specifically: mastery, self-efficacy expectancies and neuroticism in the adaptation to cardiac disease.

Mastery is a comprehensive concept of control and refers to the extent to which one assumes oneself as having control over one's life chances, unlike the fatalistic assumption that one's life is ruled by external factors (Pearlin & Schooler, 1978). Mastery is considered a key variable in adjustment (Bandura, 1977; Carver et al., 2000). Mastery is a resource that activates people in general which may increase coping efforts and persistence, providing one with a positive self-image, reducing distress and facilitating adjustment to disease (Bastone & Kerns, 1995; Mendes de Leon, Seeman, Baker, Richardson, & Tinetti, 1996). Although the beneficial role of mastery has been questioned, it has been found that mastery is related to better well-being (Thompson & Collins, 1995). Several studies among cardiac patients also describe a positive relation between mastery and psychological adjustment (Helgeson, 1999; Terry, 1992).

Self-efficacy expectancies are considered a cognitive control system that influences the likelihood of performing behaviors particularly in two situations: when new behaviors are learned, or when established behaviors are challenged (Bandura, 1977, 1982; Ewart & Fitzgerald, 1994). Since, a behavioral component may be of importance in the epidemiology of cardiac disease, self-efficacy expectancies may be particularly important in adaptation to cardiac disease. Self-efficacy expectancies were more strongly related to physical domains of health, than to psychological domains in a cross-sectional study among older persons (Kempen, Jelicic, & Ormel, 1997). When studying the predictive meaning of self-efficacy, one may distinguish global and domain-specific manifestations of self-efficacy. Some researchers have advocated the use of domain-specific measures of self-efficacy to maximize the likelihood of finding relationships in a given domain (Bandura, 1977; Ewart, Taylor, Reese, & Debusk, 1983; Lachman, 1986). However, because there were multiple domains in the present study (physical and psychological functioning), the use of a generalized measure of self-efficacy expectations is deemed more appropriate.

The construct of self-efficacy is related to mastery; they are both concepts regarding personal or perceived control. However, mastery differs from self-efficacy expectancies since mastery primarily refers to general control over one's life chances and self-efficacy relates to reaching goals. Self-efficacy includes both the belief that an outcome is achievable and the perceived capacity to produce an outcome and is typically studied as a predictor of the performance of health behavior (Grembowski et al., 1993; Menec & Chipperfield, 1997; Ziff, Conrad, & Lachman, 1995). Given the importance for the initiation of coping behaviors, both might be interesting variables to assess in relation to physical and psychological adjustment to cardiac disease.

Neuroticism is a major domain of personality that contrasts adjustment or emotional stability with maladjustment or negative emotionality (Costa & McCrae, 1980). Neuroticism includes a susceptibility to psychological distress, and also indicates a tendency to have unrealistic ideas, an inability to control urges, and inefficient ways of coping with stress (Ormel & Wohlfarth, 1991). Neuroticism has been particularly related to well being in general populations. Although, neuroticism is rarely studied as a predictor for disease adjustment, a recent study among cancer patients showed that a higher level of pre-morbid neuroticism was a significant predictor of worse psychological adjustment to cancer (Ranchor et al., 2002). A cross-sectional study among older persons showed that neuroticism was related to psychological functioning, and not to physical domains of health (Kempen et al., 1997). In addition, reductions in anxiety following cardiac rehabilitation were associated with neurotic dispositions, while improvements in activities of daily living and social activity were not (Trcieniecka-Green & Steptoe, 1994). Hence, neuroticism is predominantly found to relate to the psychological domain of health.

The described psychological attributes may influence adjustment after cardiac disease through a variety of behavioral and cognitive mechanisms (Bailis, Segall, Mahon, Chipperfield, & Dunn, 2001; Beasley, Thompson, & Davidson, 2003). One of these mechanisms relates to the process of coping (Lazarus & Folkman, 1984). The stress-coping paradigm suggests that favorable levels of psychological attributes may protect against progression of disability over time (Mendes de Leon et al., 1996; Parkes, 1986; Pearlin, Lieberman, Menaghan, & Mullan, 1981). Mastery and self-efficacy are hypothesized to influence psychological and physical functioning through the activation of individual coping processes, and may prevent or lessen stressful appraisals. Another mechanism explaining the influence of self-efficacy pertains to health behavior. For neuroticism it is hypothesized that it is related to psychological functioning through a general vulnerability mechanism, making people with higher levels of neuroticism prone to psychological dysfunction following events.

In the present study we were able to include premorbid levels of mastery, self-efficacy and neuroticism. Neuroticism, as a personality trait is considered rather stable over time (Sanderman & Ranchor, 1994), while it is debated in the literature whether levels of mastery and self-efficacy are stable over time (Lachman & Leff, 1989; Mendes de Leon et al., 1996; Rodin, 1986; Sherer et al., 1982). If indeed levels of mastery and self-efficacy expectancies may change over time, for example, as a result of serious events or therapeutic interventions, postmorbid levels of these attributes may differ from levels prior to diagnosis. Postmorbid levels may give insight into the extent to which psychological attributes are activated once needed, while premorbid levels may give an indication of the availability of the attributes (Ranchor et al., 2002). Therefore, it is of particular interest to study whether premorbid levels of psychological attributes are predictive of adjustment following disease. Such studies are scarce. Another issue concerns the influence of psychological attributes in the short-term versus effects in longer term. According to the 'Crisis Theory', an initial crisis response (in the short-term) can be distinguished from an adaptation period, involving restoring physical and psychological balance and regaining function (Moos & Schaefer, 1984). Aspects of illness and disease severity might dominate the initial crisis response to physical illness. Although an influence of psychological attributes in the short-term is not ruled out, these may be of particular importance in the longer term. The final issue, without much attention in the literature, is the combined effect of several psychological attributes. In our study, we investigate whether the effects of the three psychological attributes are independent of one another. In addition, the effects of other characteristics that may interact with adaptation to cardiac disease are included. Especially in an elderly population the effects of age, gender and comorbidity may be important features (Jaarsveld et al., 2002; Stewart et al., 1989).

The present study aims at examining the role of three psychological attributes in explaining individual differences in physical and psychological adjustment to cardiac disease in the short-term (six weeks after diagnosis) and long-term (1 year after diagnosis). Moreover, we are interested in which one of these three attributes is the most important predictor for adjustment after cardiac disease. We assume that favorable premorbid levels of the psychological attributes (high sense of mastery, self-efficacy expectancies and low neuroticism) may help patients to maintain or regain relatively high levels of physical and psychological functioning despite their cardiac disease. We hypothesize that self-efficacy expectancies and mastery affect both physical and psychological functioning, while neuroticism is in particular related to psychological functioning. In addition, we hypothesize that the effects of the three psychosocial attributes are more profound at long-term (i.e. 1 year after diagnosis) than in the short-term.

Method

This study is part of the *Groningen Longitudinal Aging Study* (GLAS). Detailed description of data collection is published elsewhere (Kempen et al., 1997; Ormel et al., 1998). Briefly, in 1993 a total of 5279 community dwelling elderly people (>56 years) were interviewed at their homes providing data on psychological attributes, determinants of disease, physical functioning, psychological functioning and well-being. Objectives, design and matters of representativeness of the GLAS study have been described elsewhere (Kempen et al., 1997). From the baseline wave in 1993 until January 1, 1998, the 27 general practitioners participating in the Morbidity Registration Network Groningen passed on the names of all patients with a new postbaseline diagnosis of cardiac disease.

Patients with a new postbaseline diagnosis of cardiac disease

Two cardiovascular events were included as indicators of cardiac disease: AMI and CHF, according to the criteria of the International Classification of Primary Care (codes K75 and K77, respectively) (Lamberts & Wood, 1987). During the enrolment period (1993–1998) 207 patients with a new episode of AMI and 293 patients with a first diagnosis of CHF after baseline were recruited. Of these 500 patients 74 (15%) died before the first follow-up assessment and 49 were already participating in one of the other six GLAS cohort studies. Of the 377 potential responders, 68 patients refused participation in the study and 47 patients did not participate for other reasons (see for details: Jaarsveld et al., 2001), leaving 262 persons for follow-up (70%). Of these 262 patients who started the follow-up study, 208 (79%) completed follow-up. These 208 patients (89 AMI and 119 CHF) were the participants of this study. Dropout during follow-up was in 20 of the 54 patients due to death. Response was 42% of the 500 original diagnosed individuals. Non-response analyses showed that at baseline, participants were significantly younger and had better physical functioning scores, on average, than non-participants. No significant differences between participants and non-participants were found on other sociodemographic variables, on psychological attributes or psychological functioning at baseline.

Assessment points

The analyses include three assessment points: baseline (premorbid), six weeks after diagnosis, and 1 year after

diagnosis. The premorbid assessment took place in 1993 for all participants, while the timing of follow-ups depended on the time of diagnosis and occurred between 1993 and 1999. The mean period between baseline and cardiac diagnosis varied from 1 to 58 months, with a mean length of 26 months (SD = 15).

Outcome measures

The outcome measures were assessed at all three assessment points, including one premorbid and two postmorbid assessments. Data on physical functioning were collected at the home of the respondent with face-to-face interviews by well-trained middle-aged women while data on psychological functioning were collected by self-report questionnaires.

Physical functioning was assessed with the *Groningen Activity Restriction Scale* (*GARS*). The GARS comprises 18 activities of daily living (ADL) and instrumental activities of daily living (IADL) items. Examples of GARS items are "Can you dress yourself without any help from others?", and "Can you walk up and down the stairs?". Each item has four answer options: (1) "Yes, I can do it fully independently without difficulty" (2) "Yes, I can do it fully independently but with some difficulty"(3) "Yes, I can do it fully independently but with great difficulty" and (4) "No, I cannot do it independently, I can only do it with someone's help". Scores may range from 18 (no physical dysfunctioning) to 72 (maximum level of physical dysfunctioning). The results of previous studies showed that GARS meets the stochastic cumulative scalability criteria of the Mokken Model, indicating the hierarchical character of the scale (Kempen, Miedema, Ormel, & Molenaar, 1996). The internal reliability estimate was .90 at baseline for the present sample.

Depressive symptoms and feelings of anxiety, indicators of *psychological functioning*, were assessed with the *Hospital Anxiety and Depression Scale (HADS)* (Zigmond & Snaith, 1983; Spinhoven et al., 1997). The HADS depression subscale was originally developed to reveal possible depressive states in a medical outpatient clinic setting. Items referring to symptoms that may have a physical cause (e.g. insomnia and weight loss) are not included in the scale. Therefore, HADS is considered to have no bias towards depressive symptoms resulting from concurrent general medical conditions (Spinhoven et al., 1997). Examples of items are "I still enjoy the things I used to enjoy", "I feel cheerful" and "I get sudden feelings of panic". Each item has four answer options. Both subscales consist of seven items and the theoretical ranges vary from 0 to 21; higher scores indicate more symptoms. HADS has been validated for an older Dutch population (Spinhoven et al., 1997). The internal reliability estimates for depressive symptoms was .73 and for anxiety .84 at baseline for the present sample.

Premorbid psychological attributes

The three psychological attributes were measured at baseline in 1993, i.e. before the diagnosis of cardiac disease, using self-report questionnaires.

Mastery was assessed with a seven-item scale (Pearlin & Schooler, 1978). This scale assesses global beliefs regarding one's ability to control an event versus being controlled by fate. Examples of mastery items are "I have little control over things that happen to me", "There is really no way I can solve some of the problems I have", "There is little I can do to change many of the important things in my life". The items are rated on a five-point Likert scale ranging from "strongly agree" to "strongly disagree" and summed to a total score, which may range from 7 to 35. Higher scores indicate stronger beliefs of mastery.

Self-efficacy expectancies was assessed with a 16-item scale developed by Sherer and adapted by Bosscher (Sherer et al., 1982; Bosscher, Smit, & Kempen, 1997). Examples of the self-efficacy expectancies items are "When I set important goals for myself I rarely achieve them", "I avoid facing difficulties" and "When trying to learn something new, I soon give up if I am not initially successful". The items are rated on a five-point Likert scale ranging from "strongly agree" to "strongly disagree" and summed to a total score that may range from 16 to 80. Higher scores indicate stronger beliefs that one has the ability to perform a desired action.

Neuroticism (or emotional instability) was assessed using a 12-item subscale of the revised version of the Eysenck Personality Questionnaire, EPQ-R) (Eysenck, Eysenck, & Barrett, 1985). Examples are "Does your mood often go up and down?", "Do you ever feel 'just miserable' for no reason?", and "Are your feelings easily hurt?" Each item has two answer categories: yes or no. Total scores may range from 0 to 12. Higher scores indicate higher levels of neuroticism.

The psychometric properties of the Dutch versions of the three scales have been assessed as satisfactory in previous (pilot) studies (Bosscher et al., 1997; Kempen, 1992; Sanderman, Arrindell, Ranchor, Eysenck, & Eysenck, 1995). Test–retest reliability correlations were available for mastery (.67 at 8 weeks) and neuroticism (.78 at 1 year). The internal reliability estimates in the present sample at baseline were .74 (mastery), .85 (self-efficacy expectancies) and .83 (neuroticism), respectively.

Covariates

Gender, age, comorbidity, disease severity and baseline (premorbid) functioning were found to be related with cardiac disease outcome in the present data and in other studies (Penninx et al., 2001). Comorbidity was included as a covariate since patients with more than

one chronic condition showed significantly greater decrements in functioning than patients with only one chronic condition (Stewart et al., 1989). Comorbidity was assessed at baseline according to the number of chronic conditions using a self-report questionnaire (Berg & Bos, 1989). Participants were asked whether they suffered from one or more of 19 chronic medical conditions in the 12 months prior to the baseline interview. In order to reduce report-bias, only those conditions that require GP or specialist consult and/or prescription of medicine were counted. Disease severity was assessed according to the New York Heart Association (NYHA) classification at the first follow-up assessment (six weeks after diagnosis) using a self-report questionnaire. This NYHA classification indicates the severity of cardiac symptoms by documenting the level of complaints of breathlessness in relation to physical activities, and may range from I (mild symptoms) to IV (severe symptoms) (New York Heart Association, 1964). Adjustment to cardiac disease might be associated with diagnosis (AMI versus CHF) or the length of the interval between baseline and the first follow-up assessment (six weeks after diagnosis). Only those potential covariates that were found to be related to the psychological attributes were included in the analyses. The assessment of comorbidity and disease severity were both self-report during a face-to-face interview.

Analyses

The relationships between each of the three psychological attributes, covariates and premorbid levels of functioning were examined using Pearson correlations (two-tailed significance). A series of hierarchical multiple linear regression analyses were conducted to examine the effects of the three psychological attributes on physical and psychological adjustment. The effect of the three psychological attributes on physical and psychological functioning at six weeks (first set of regression analyses) and at 1 year (second set) after diagnosis is examined, adjusting for levels of premorbid (baseline) functioning and covariates (age, gender, comorbidity, disease severity). In all regression models variables are entered in the following order: functioning at baseline (step 1), covariates (step 2), psychological attributes (step 3). For both sets of regression analyses the effects of each attribute separately is examined as well as a combined effect of all three attributes to study whether effects are independent and which of the attributes is most important.

Before the regression analyses were conducted, the outcome variable physical functioning (GARS) was transformed logarithmically. As a result the skewness coefficients were reduced from 2.0 to 1.2 for baseline scores, and from 1.1 to 0.5 for scores at six weeks and 1 year, which were considered as acceptable. The variance inflation factors (VIF) for the predictors were checked for multicollinearity. All VIF scores were much lower than 10.0, which can be considered as acceptable. Results were considered significant if $p < .05$. Data were analyzed using SPSS/PC software, version 11.

Results

Analyses included 208 patients. Patient characteristics are shown in Table 1. Mean age at baseline is 71.9 years (SD = 7.8), indicating a relatively elderly population. At baseline, only 48 patients (23%) report to have no chronic condition, while on average patients report to have 1.6 chronic conditions prior to cardiac diagnosis. At baseline, 36% of our sample reported a heart condition (other than AMI or CHF) and 25% reported to have hypertension. The prevalence of other chronic disease at baseline is: asthma or chronic bronchitis (15%), back problems for at least three months or slipped disc (14%), joint conditions or arthritis (14%), diabetes mellitus (11%). Other diseases such as dermatological or gastric disorders occurred in less than 10% of patients. Table 1 also includes descriptive statistics of the psychological attributes and functioning at each assessment point. Following diagnosis, an increase in physical dysfunction, depressive symptoms and anxiety is observed at the group level, with large individual differences as indicated by the range and SD.

Table 2 presents the interrelations between the three psychological attributes, covariates and premorbid functioning. Although mastery and self-efficacy expectancies are significantly interrelated, $r(205) = .53, p < .001$, they represent different concepts. Neuroticism is significant negatively related to mastery, $r(205) = -.42, p < .001$, and self-efficacy expectancies, $r(205) = -.37, p < .001$. High levels of both mastery and self-efficacy expectancies are related to lower age, and male gender; in addition high mastery is related to less comorbidity and less severity of the disease; high neuroticism is related to lower age, increased comorbidity as well as to disease severity. Therefore, these covariates may contribute to differences in physical and psychological adjustment to cardiac disease and are included in the analyses. The specific diagnosis (AMI versus CHF) and the length of the interval between baseline and the first follow-up assessment are not significantly correlated to the psychological attributes (data not shown). In addition, diagnosis and interval are non-significant covariates in the multivariate analyses and outcomes are unaffected by these variables. Therefore, diagnosis and interval are not included as covariates in the final analyses.

Table 3 comprises the outcomes of the multiple regression analyses for *physical functioning*. Models B,

Table 1
Descriptive statistics of the study sample ($N = 208$)

	%	*M*	(SD)	Range
Age (yrs)		71.9	(7.8)	58–96
Nr. of comorbid chronic conditions		1.6	(1.4)	0–7
Interval between baseline and cardiac diagnosis (months)		26.4	(15.4)	1–58
Gender				
Females	48			
Males	52			
Diagnosis				
CHF	57			
AMI	43			
NYHA classification				
Class I	32			
Class II	19			
Class III	39			
Class IV	10			
Mastery		23.8	(4.8)	11–35
Self-efficacy expectancies		58.4	(11.6)	23–80
Neuroticism		3.8	(3.2)	0–12
Physical functioning				
at baseline		24.1	(8.0)	18–59
at 6 weeks		28.5	(9.8)	18–61
at 1 year		29.2	(10.3)	18–63
Depressive symptoms				
at baseline		5.1	(3.8)	0–18
at 6 weeks		5.0	(3.6)	0–17
at 1 year		5.8	(4.1)	0–19
Feelings of anxiety				
at baseline		3.9	(3.7)	0–17
at 6 weeks		4.9	(3.8)	0–17
at 1 year		5.2	(4.2)	0–21

C and D show significant effects of all three psychological attributes after adjusting for baseline functioning, age and NYHA class. Worse physical functioning at six weeks is predicted by worse physical functioning at baseline, higher age, more severe disease, low levels of mastery and self-efficacy expectancies and high levels of neuroticism. The final model (E) combining all attributes shows an independent effect of self-efficacy expectancies for physical functioning at six weeks. Although, the effect of self-efficacy expectancies is significant, it only adds 2% to the explained variance of model (A) including baseline functioning and covariates ($R^2 = .62$). Models B–E for physical functioning at 1 year show a significant effect of self-efficacy expectancies. The long-term effect of self-efficacy expectancies is again small (R^2 change is 1%) but statistically significant. The final models (E) show that, the effect of self-efficacy expectancies on both short- and long-term physical adjustment goes beyond mastery and neuroticism.

Table 4 shows the results for *depressive symptoms*. Models B–D show significant effects of all three psychological attributes after adjusting for baseline symptoms and NYHA class. Higher depression scores at six weeks are predicted by high depression scores at baseline, more severe disease, low levels of mastery and self-efficacy expectancies and high levels of neuroticism. The final model (E) combining all attributes shows independent effects of self-efficacy expectancies and neuroticism, on the short-term. The psychological attributes add a significant 5% to the explained variance, resulting in a total of 37% explained variance. The models (B–D) for depressive symptoms at 1 year show significant effects of all three attributes. The final model (E) combining all attributes shows independent effects of mastery and neuroticism for depression at the long-term, these add 6% explained variance to the original model (A).

Table 2
Pearson correlations between the psychological attributes, covariates and baseline (premorbid) functioning[a]

	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Mastery	+.53***	−.42***	−.20**	−.19**	−.19**	−.17*	−.42***	−.50***	−.47***
2. Self-efficacy expectancies	—	−.37***	−.19**	−.19**	−.11	−.13	−.25***	−.39***	−.38***
3. Neuroticism		—	−.14*	+.07	+.14*	+.20**	+.13	+.45***	+.65***
4. Age			—	+.23**	+.12	+.10	+.37**	+.13	−.02
5. Gender (1 = ♂, 2 = ♀)				—	+.17*	+.13	+.30**	+.12	+.23**
6. Comorbidity					—	+.28***	+.38***	+.21**	+.27***
7. NYHA[a]						—	+.25**	+.17*	+.13
8. Premorbid physical functioning							—	+.26***	+.24**
9. Premorbid depressive symptoms								—	+.59***
10. Premorbid level of anxiety									—

*$p < .05$,**$p < .01$,***$p < .001$.
[a]Variables are assessed at baseline (premorbid) except for NYHA, which is assessed 6-weeks postmorbid.

Table 3
Regression models for physical functioning at follow-up; numbers are standardised beta coefficients[a]

Regression model		A	B	C	D	E
Physical functioning at 6 wk	Phys. funct. at baseline	.60***	.56***	.57***	.58***	.57***
	Age	.24***	.23***	.22***	.25***	.22***
	NYHA class	.19***	.19***	.18***	.17***	.18***
	Mastery		−.10*			n.s.
	Self-efficacy exp.			−.15**		.15**
	Neuroticism				.09*	n.s.
Model R^2		.62	.63	.64	.63	.64
Physical functioning at 1 yr	Phys. funct. at baseline	.60 ***	.58 ***	.58 ***	.59 ***	.58***
	Age	.22 ***	.21 ***	.20 ***	.25 ***	.20***
	NYHA class	.20 ***	.19 ***	.18 ***	.19 ***	.18***
	Mastery		−.07	−.13**		n.s.
	Self-efficacy exp.				.06	−.13**
	Neuroticism					n.s.
Model R^2		.62	.62	.63	.62	.63

Model A: Physical functioning at baseline and age, sex, comorbidity, NYHA class (only significant covariates are shown in table).
Model B: Model A + Mastery.
Model C: Model A + Self-efficacy expectancies.
Model D: Model A + Neuroticism.
Model E: Model A + Mastery, self-efficacy expectancies and neuroticism (stepwise method).
*$p<.05$,** $p<.01$,*** $p<.001$.
[a]Logarithmic transformed scores of physical functioning are used in this analyses.

Table 4
Regression models for depressive symptoms at follow-up; numbers are standardised beta coefficients

Regression model		A	B	C	D	E
Depression at 6 wk	Depression at baseline	.50***	.43 ***	.44***	.43***	.39***
	NYHA class	.21***	.19 **	.19**	.19**	.18**
	Mastery		−.17*			n.s.
	Self-efficacy exp.			−.19**		−.16*
	Neuroticism				.17**	.15*
Model R^2		.32	.35	.36	.34	.37
Depression at 1 yr	Depression at baseline	.44***	.32***	.37***	.39***	.27***
	Age	.15*	.12*	.12*	.20***	.16*
	NYHA class	.17**	.15*	.15*	.14*	.13*
	Mastery		−.25***			−.20**
	Self-efficacy exp.			−.21**		n.s.
	Neuroticism				.22**	.17*
Model R^2		.28	.32	.31	.31	.34

Model A: Depressive symptoms at baseline and age, sex, comorbidity, NYHA class (only significant covariates are shown in table).
Model B: Model A + Mastery.
Model C: Model A + Self-efficacy expectancies.
Model D: Model A + Neuroticism.
Model E: Model A + Mastery, self-efficacy expectancies and neuroticism (stepwise method).
*$p<.05$, ** $p<.01$, *** $p<.001$.

Table 5 shows the results for *feelings of anxiety*. Models B–D show only a significant effect of neuroticism for anxiety levels at six weeks. High levels of anxiety at baseline, lower age, more severe disease and high levels of neuroticism predict higher levels of anxiety at six weeks. Neuroticism adds a significant 4% to the explained variance of model A. The final model (E) explains 33% of the variance. The models B–D for

Table 5
Regression model for feelings of anxiety at follow-up; numbers are standardised beta coefficients

Regression model		A	B	C	D	E
Anxiety at 6 wk	Anxiety at baseline	.39***	.33***	.37***	.21**	.21**
	Age	−.18**	−.20**	−.19**	−.14*	−.14*
	NYHA class	.29***	.28***	.29***	.26***	.26***
	Mastery		−.13			n.s.
	Self-efficacy exp.			−.06		n.s.
	Neuroticism				.29***	.29***
Model R^2		.29	.29	.28	.33	.33
Anxiety at 1 yr	Anxiety at baseline	.50***	.43***	.46***	.35***	.30***
	NYHA class	.18**	.16**	.17**	.15*	.14*
	Mastery		−.17**			−.14*
	Self-efficacy exp.			−.12		n.s.
	Neuroticism				.23**	.22**
Model R^2		.30	.32	.31	.33	.34

Model A: Feelings of anxiety at baseline and age, sex, comorbidity, NYHA class (only significant covariates are shown in table).
Model B: Model A + Mastery.
Model C: Model A + Self-efficacy expectancies.
Model D: Model A + Neuroticism.
Model E: Model A + Mastery, self-efficacy expectancies and neuroticism (stepwise method).
*$p<.05$, **$p<.01$, *** $p<.001$.

anxiety levels at 1 year show significant effects of mastery and neuroticism. The final model (E) combining all attributes shows independent effects of mastery and neuroticism for anxiety at the long-term (R^2 change is 4% resulting in 34% for the total model).

Discussion

The results of this prospective longitudinal study showed that premorbidly assessed levels of mastery, self-efficacy expectancies and neuroticism were significant predictors of adjustment after cardiac disease. The influence of the psychological attributes differed to some extent with time and domain (physical versus psychological adaptation). Multivariate analyses show that mastery affected only psychological adaptation (depressive symptoms and anxiety) in the longer term. Self-efficacy expectancies affected physical adaptation in the short- and long-term as well as depressive symptoms in the short-term. Neuroticism affected psychological adaptation (depressive symptoms and anxiety) in the short- and longer term.

These multivariate analyses indicated whether the three attributes had additive effects on outcome, or whether their effects overlapped. This is relevant since the attributes were significantly interrelated. Judge, Erez, Bono, and Thoresen (2002) argued that neuroticism and self-efficacy expectancies are markers for the same higher concepts, however they found independent effects of neuroticism and self-efficacy expectancies.

The data show that prior physical functioning, age, and disease severity explain a large part of the variance in physical functioning at six weeks and at 1 year (62%). In the short- and long-term, self-efficacy expectancies add significant to the model, by increasing the explained variance with 1–2%. These effects are statistically significant, however their small magnitude calls for prudence. The observed effects and clinical impact may have been limited in our study since a generalized measure of self-efficacy was used. The use of a generalized measure was deemed more appropriate in our study since multiple domains (physical and psychological functioning) were studied and assessment took place prior to the cardiac event. Taking this into account, the observed significant effects indicate the relevance of self-efficacy expectancies. Moreover, a more focussed measure of self-efficacy may exert a greater impact on adjustment.

As for psychological adaptation, prior levels of depression or anxiety, age and disease severity explain 28–32% of the variance in psychological functioning at follow-up. Neuroticism significantly adds to all models for psychological functioning, and self-efficacy and mastery are alternately related to psychological functioning. As summarized in the Introduction, the literature emphasizes an effect of neuroticism on psychological domains of health, while mastery and self-efficacy expectancies were assumed to affect both domains of health. The data show that the effects for self-efficacy expectancies contribute to both physical and psychological adjustment. While neuroticism and

mastery particularly relate to psychological adjustment, the influence of mastery and neuroticism on physical adjustment does not go beyond that of self-efficacy expectancies. The predominant influence of self-efficacy expectancies on physical adjustment may be related to the focus of self-efficacy expectancies on reaching goals and the perceived capacity to produce outcome, in contrast to mastery, which primarily relates to general control over one's life chances. These findings are relevant in the discussion about the difference between mastery and self-efficacy expectations. Although both concepts are assessed at a general level in this study, they are found to have differential effects on either physical or psychological adjustment. In addition, we hypothesized that the effects of the psychological attributes were more profound at long-term (i.e. 1 year after diagnosis) than in the short-term. Our data do not support this. The psychological attributes both in the short and the longer terms predict adjustment.

As mentioned in the Introduction, the inclusion of premorbidly assessed psychological attributes in our study is unique and has clear implications. Persons with relatively unfavorable levels of psychological attributes are at increased risk for maladjustment after cardiac disease. Since attributes are assessed before the cardiac diagnosis, the assessment is not biased by a possible influence of the cardiac diagnosis. In addition premorbid levels of physical and psychological functioning are included in our analyses. By taking into account these premorbid levels of functioning, we are able to study adjustment to cardiac disease.

Some comments have to be made regarding these results. Due to several reasons (see Method) we included 208 persons in our study out of the 500 originally diagnosed. However, unlike other studies in this field, we were able to cover all incident cases in a selected sample, including those with a poor prognosis. It is likely that this produces the seemingly high non-response rates. Many longitudinal studies make restrictions with respect to the prognosis of patients by including only patients who are likely to survive during the study period. The reasons for actual non-response show that non-participants were older and had poorer physical functioning compared to the participants, and this may have affected our outcomes. The results concern a relatively healthy subset of patients. This is not necessarily a shortcoming since the issue of psychological predictors for adjustment is by definition only of relevance in patients who survive the disease for at least that period. Our conclusions regarding the relevance of psychological attributes for future adaptation to cardiac disease are therefore limited to the relatively less affected patients who survive at least a year.

In this study two frequently occurring cardiac diseases (CHF and AMI) are studied together. However, the adaptation to these cardiac diseases might differ. Disease specific analyses of our data (with less power) showed however roughly the same results. An earlier paper examined the influence of the same psychological attributes on physical decline in the first six weeks after diagnosis, and found unique contributions of self-efficacy expectancies for physical decline after CHF, while mastery significantly contributed to physical decline after AMI (Kempen, Sanderman, Miedema, Meyboom-de Jong, & Ormel, 2000). Disease specific analyses in the present paper did not confirm this for physical decline on the long-term, or for psychological adjustment. In addition, the time interval between the premorbid assessment and cardiac diagnosis varied from immediately after baseline to 58 months ($M = 26$ months, $SD = 15$). The influence of psychological attributes to physical and psychological adaptation might change overtime. The outcomes of our analyses hardly changed when the baseline to cardiac diagnosis interval was included as covariate in the multivariate models.

To conclude, this longitudinal study showed that psychological attributes do have a role in explaining individual differences in vulnerability to negative consequences of cardiac disease. Neuroticism is a recurring attribute for psychological adaptation to cardiac disease. Therefore, cardiac patients with a relatively higher level of neuroticism may be at risk for hampered psychological adaptation, suggesting that these patients might benefit from early psychological interventions. The significant influences of mastery and self-efficacy expectancies on adaptation might also be of clinical importance. It is debated in the literature whether changes in generalized, more global senses of mastery and self-efficacy occur among elderly people, while changes in domain specific measures are plausible (Lachman & Leff, 1989; Mendes de Leon et al., 1996; Sanderman & Ranchor, 1994). Further research is necessary to study whether it is possible to increase levels of these attributes by intervention programs. If, indeed mastery and self-efficacy expectancies could be increased by interventions in patients with low levels, this might be clinically relevant for adaptation to cardiac disease. In clinical practice, this implies that special attention is warranted in patients with low self-efficacy, low mastery and high neuroticism, and efforts to increase self-efficacy or mastery or to prevent declines in these attributes may be important.

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